

Divergent evolution paths of different genetic families in the Penna model

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Abstract. We present some results of simulations of population growth and evolution, using the standard asexual Penna model, with individuals characterized by a string of bits representing a genome containing some possible mutations. After about 20000 simulation steps, when only a few genetic families are still present from among rich variety of families at the beginning of the simulation game, strong peaks in mutation distribution functions are observed. This known effect is due to evolution rules with hereditary mechanism. The birth and death balance in the simulation game also leads to elimination of families specified by different genomes. Number of families $G(t)$ versus time t follow the power law, $G \propto t^n$. Our results show the power coefficient exponent n is changing as the time goes. Starting from about -1 , smoothly achieves about -2 after hundreds of steps, and finally has semi-smooth transition to 0 , when only one family exists in the environment. This is in contrast with constant n about -1 as found, for example, in [1]. We suspect that this discrepancy may be due to two different time scales in simulations - initial stages follow the $n \approx -1$ law, yet for large number of simulation steps we get $n \approx -2$, providing random initial population was sufficiently big to allow for still reliable statistical analysis. The $n \approx -1$ evolution stage seems to be associated with the Verhulst mechanism of population elimination due to the limited environmental capacity - when the standard evolution rules were modified, we observed a plateau ($n = 0$) in the power law in short time scale, again followed by $n \approx -2$ law for longer times. The modified model uses birth rate controlled by the current population instead of the standard Verhulst death factor.

Introduction

The Penna model [2] is a one of variety of mutation accumulation models. They are based on assumption that biological ageing is caused by deleterious mutations in genome (it is inherited from parent – asexual model – and enriched by additional mutations put at random site at genome at the birth moment).

The number of genetic families in the population decreases as the time grows. This was described for example in papers [2], [3]. The effect of the reduction in

number of genetic families is also responsible for sharp and irregular maxima in the mutation distribution function after many time steps of computer simulation. This effect is discussed in the main text.

Model

In the model presented, individuals genome is represented as a string of bits, where '1' means presence of mutation, and '0' – lack of mutation. When an individual reaches age a , only the first a bits are exposed. The number of “ones” in that part of genome are considered as active mutations m . When the number m of active mutations exceeds a threshold T , $m \geq T$ (one of the model parameters) – the individual dies due to genetic reason. Another important parameter is R – minimal reproduction age – only individuals with $a \geq R$ can give offspring. That causes some genomes not to be reproduced, because its owner dies before reaching mature age. This is the basic implementation of Darwinian evolution mechanism.

There is also another reason of death incorporated into the model – death due to overpopulation – controlled by so called Verhulst factor. The probability of such death equals to $n(t)/N$, where $n(t)$ is number of individuals in time step t , and N is the environmental capacity (which is constant during the simulation). This factor limits the population size.

We start the simulation with population which has no mutations (genome length is 32 bits). It takes several hours using Pentium III class machine. In each time step:

- each individual is tested against possible death, due to:
 - genetic reason, when $m \geq T$;
 - Verhulst factor with probability $n(t)/N$;
- if individual survived and reached age R , it gives birth to B babies on average, so B is the birth rate. Each descendant gets the genome inherited from parent, enriched by M new mutations. (If one of the M mutation attempts hits an already mutated bit, this bit stays as it is.)
- all survivals and newly born individuals make up population at the next time step.

Population structure

By a population structure we mean set of parameters, we use to characterize it. The first one is the survival factor:

$$s(a, t) = \frac{n(a+1, t+1)}{n(a, t)}, \quad (1)$$

where $n(a, t)$ is number of individuals of age a at time t . This survival factor is interpreted as a probability that individual which has age a , enters the next

time step $t + 1$. From this – we can obtain mortality rate:

$$q(a, t) = 1 - s(a, t) = 1 - \frac{n(a + 1, t + 1)}{n(a, t)} \quad (2)$$

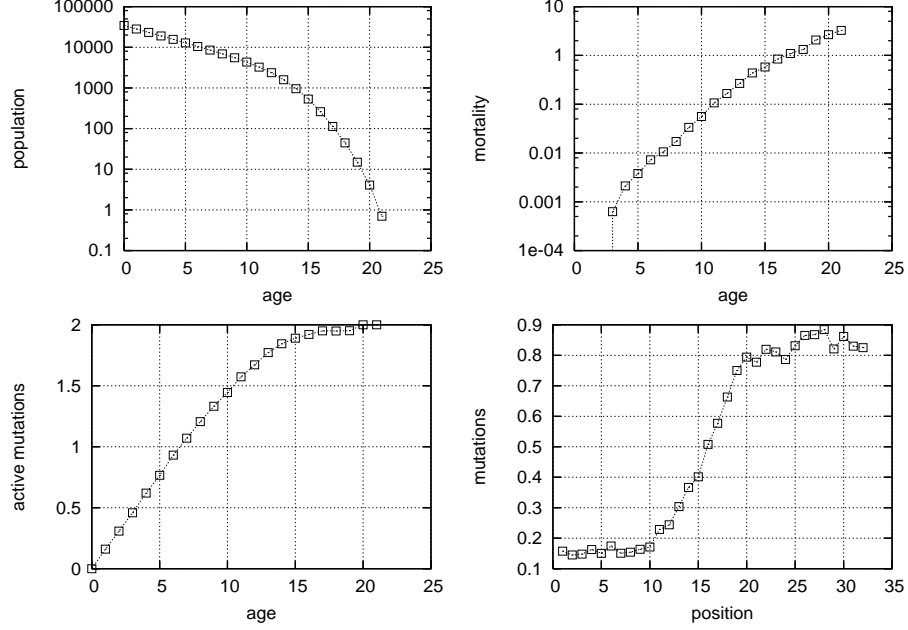


Fig. 1. Population structure after 1000 steps. Standard Penna model $(N, B, R, M, T) = (10^6, 1.0, 7, 1.0, 3)$.

We can separate mortality for two elements – mortality because of Verhulst factor, and mortality because of achieving threshold T by number of active mutations:

$$q(a, t) = q_v(a, t) + q_g(v, t) = \frac{v(a, t) + g(a, t)}{n(a, t)}, \quad (3)$$

where $v(a, t)$ is number of individuals killed because of Verhulst factor, and $g(a, t)$ is number of deaths caused by genetic reason. The quantity $q_v(a, t) = q_v(t)$ is age-independent, whereas $q_g(a) = \lim_{t \rightarrow \infty} q_g(a, t)$ gives approximately dependency:

$$q_g(a) \approx q_0 \cdot \exp(a \cdot b), \quad (4)$$

(where q_0 and b are constants) which is known as Gompertz law [1]. It is an empirical law observed also in human population (see [4], [5]).

Figure 1 presents results after 1000 time-steps of simulation (averages from last 20 steps), when the average number of births equals average numbers of

deaths. As can be seen at the chart in the right top corner – Gompertz law is fulfilled (it presents $q_g(a)$). The chart at the right down corner, presents the average number of mutations versus bit position. Low values at the beginning are the effect of evolution mechanism, that eliminates genotypes with big number of mutations at this part of genome (individuals with such genome can not reproduce because they die before reaching age R). As it can be seen in the next section, at this case the population is quite diverse and the result significantly differs from that after 20000 steps (the same parameters, figure 2).

Decline of a number of genetic families

The population characteristics presented in the previous section (after 1000 steps) could lead us to misleading conclusions. Average number of birth acts and average number of deaths are equals (and what follows – average population size does not change), so it may look like a stationary state. Indeed, it is so in terms of the population size, however the evolution is still shifting the genome distribution function. The proportions of participation of particular genotypes in population are changing. It is so since first we need to define the criterion with respect to which the close-to-stationary state is claimed. If the total population $n(t)$ is considered, one hundred iterations may be enough. However if we mean overall characteristics of genomes, it is far from the equilibrium and we must proceed simulation for many thousands steps. There are different time scales for different features observed in population.

To distinguish between different genotypes – we introduced a special marker of individuals. At the beginning of simulation – each individual inserted into environment, has given unique number. When individual gives birth – the marker is inherited by progeny. The marker is kept fixed which makes it possible to trace the evolution of different genetic families (by genetic family we mean set of individuals with the same marker).

Figure 2 presents results of simulation after 20000 steps (this is the continuation of simulation presented at the previous section). Peaks in mutations distribution – the right down corner – suggest that the population became more homogeneous. To check our hypothesis we plotted the number $G(t)$ of genetic families versus the time step – full squares at figure 3 (double log scale). As can be seen it diminishes rapidly – and after about 500 steps becomes a power function of time.

In our case – starting with as many as $4 \cdot 10^5$ genetic families, after about 20000 steps we got three families in environment. For a long time run we get results presented in figure 4.

As it was mentioned earlier, we observe that $G(t)$ dependency is:

$$G(t) \sim t^n \tag{5}$$

It seems to be independent on Penna model parameters as long as we not approach to the logistic model – empty squares at figure 3 (by logistic model, we mean model where the only reason of death is overpopulation, no genetic factor

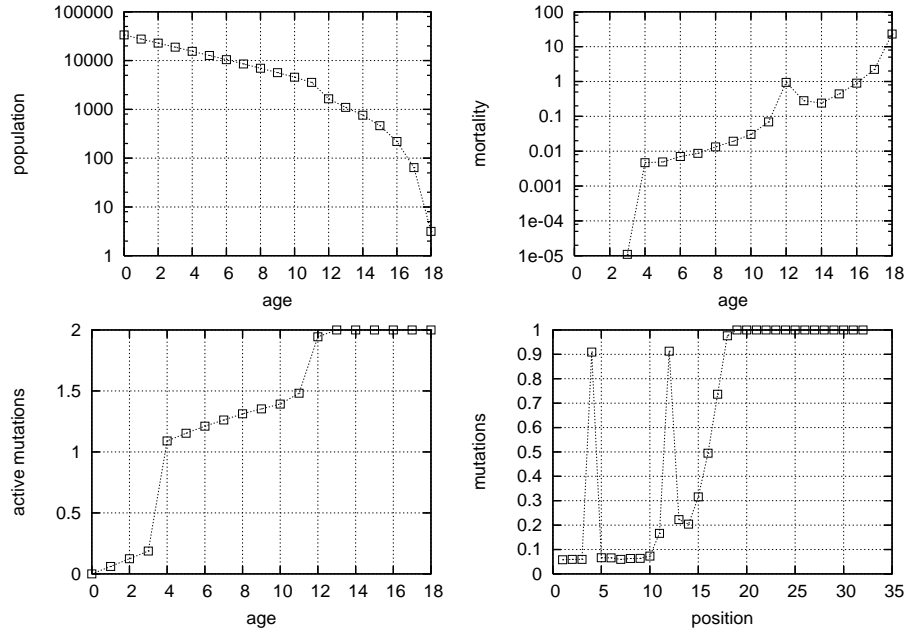


Fig. 2. Population structure after 20000 steps. Standard Penna model $(N, B, R, M, T) = (10^6, 1.0, 7, 1.0, 3)$. Strong peaks in mutations distribution (result of Eve effect) is also reflected in the mortality $q(a)$ case.

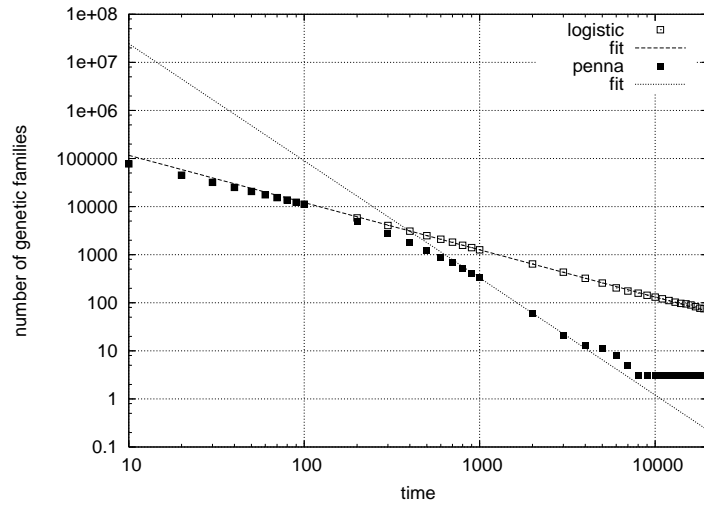


Fig. 3. Number of genetic families in environment as a function of time. Full squares – Penna model, empty squares – logistic model.

is present). This result is compatible with results described in [1] but with one exception. In our simulations $n \approx -2$ for asexual model ($n \approx -1$ for logistic one), while in literature $n \approx -1$ is claimed. We suppose that this discrepancy is caused by the fact, that we start simulation with much bigger initial population, and so we have had a chance to achieve the $n \approx -2$ region, that is before the population is extinct.

Our three families as the final result, is rather an effect of under-counting the simulation. If the simulation time is sufficiently big, the number of families achieves 1 every time – see figure 4. This phenomena is known as “Eve effect”. What is more, the $G(t)$ curve achieves $n \approx -1$ region again at the end. So there are three stages of that play: -1 from the start, to several hundred time-steps, then -2 until time reaches about 10000 steps, and final -1 (figure 4).

We would like to emphasize, that whole “s-shape” effect does not occur in sexual model. In that case – whole curve is without any transitions, and has an exponent equals -1 (what remains in accordance with results presented in [1]).

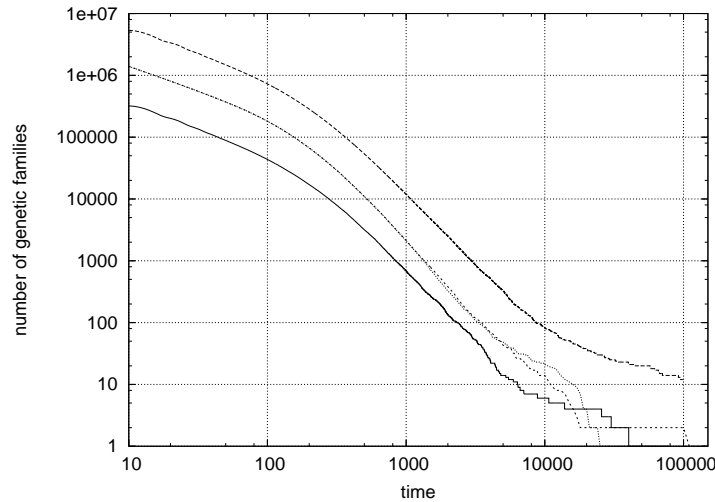


Fig. 4. Number of genetic families versus time – four different simulations. The most top curve (dashed) and the most bottom (solid) are obtained from results given us by Dietrich Stauffer [6] (for initial population equals accordingly 10^7 and $6 \cdot 10^5$). Two curves in the middle (dotted and short dashed) originate from our simulations – all parameters are the same in this two cases except pseudo-random number generator initial value (initial population equals $2 \cdot 10^6$).

We suppose that an important role in the presented “Eve effect” plays the Verhulst factor. This is the only way which one family can influence on another. When one family accidentally outnumbers another – it can produce more new individuals filling up the environment, and death because of overpopulation makes

not as big damage as in less numerous families. Interesting could be the introduction of some mechanism into the model, that allows for exchanges between families which may change the final result.

Modified Penna model - with no Verhulst factor

Different mechanisms leading to elimination of individuals gives insight to the evolution game. The Verhulst factor, however, was introduced in the past as a remedy to avoid unlimited growth of population. It was not devised to account for specific death cause such as disease, genetic death, hunting etc. When we go for more detailed modeling, one should replace the overall Verhulst factor by more specific mechanisms. Some papers are devoted to this problem. The deterministic case of two families is discussed in [7], and Verhulst factors are completely removed in [8].

We explore modification introduced in [9] – a dynamic version of birth rate which helps to keep the size of the population stable and finite. The birth rate changes in each time-step according to rule:

$$B(t) = \begin{cases} B_c \cdot (1 - \frac{n(t)}{N}) & \text{for } n(t) \leq N \\ 0 & \text{for } n(t) > N \end{cases} \quad (6)$$

where B_c is constant (set at the beginning of simulation). The next change is an elimination of the Verhulst factor as a possible cause of death – individuals death occurs only because of genetic reasons. A comparison of results of the two simulation – standard and modified Penna model – is presented in figure 5

Simulations were done for the same set of parameters as presented before, except $N = 610000$ for the modified model (matched to get comparable results) and $B_c = 1$. As can be seen, there is a *plateau* at the initial time range in $G(t)$ dependency, in the modified model. At the rest of time range – dependencies are similar. The Eve effect is still present. It seems, that replacing Verhulst factor by dynamic birth rate does not change this fact.

Conclusions

The early stage simulation, up to about several hundred steps, corresponds to the phase of already established size of the population and still far from the stationary bad mutation distribution (at least for the assumed rate of the bad mutations). Then the genetic death rate is not a dominant factor as yet, and so the model follow the rules more as if the logistic scheme which ignores genetic death factor only. That is why we may expect the $n = -1$ coefficient as predicted by the logistic model.

When continuous pumping in the bad mutations yields a sort of saturation in the bad mutation accumulations for simulation lasting long enough (say 1000 steps or so), the genetic death may prevail and we are inclined to associate the $n = -2$ coefficient with this region.

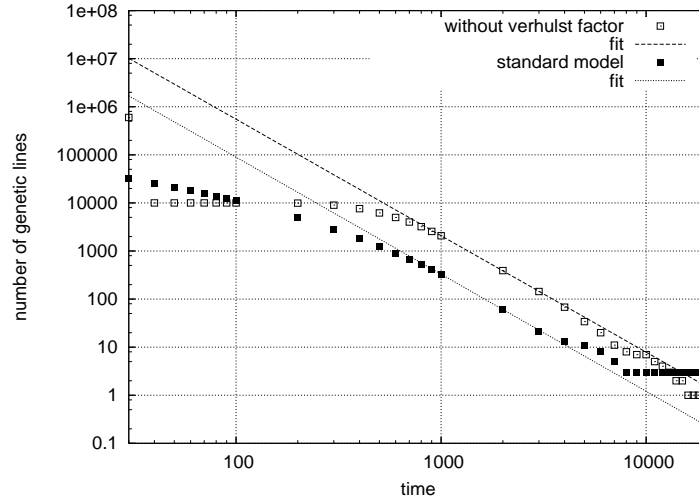


Fig. 5. Number of genetic families in environment as a function of time. Full squares – standard Penna model, empty squares – Penna model without Verhulst factor

Unavoidably, however we approach the final equilibrium with one family only. Obviously this very final stage is $n = 0$ and so we may anticipate a smooth transition from $n = 2$ to $n = 0$ when the number of families drop down to small number of a few competing families.

The one only final family confirms the known effect of marginal stability of different families, when competing in a limited environment in the logistic model. Once the genetic death is switched on (like in the Penna-like model), the competition leads to elimination of the weaker groups. The time scale of this process is of order of 10^5 steps, so we need to carry on the many simulation steps before finally the Eve emerges as the only representative family of the whole population.

The last section on modified Penna model proves that the value of the exponent n in the power law $G(t) \propto t^n$ is indeed model-dependent. In short, the Penna model with birth rate controlled by the current size of the population leads to plateau $n = 0$, asexual Penna model yields $n = -1$, at the beginning several hundred of steps.

Acknowledgments

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We would like to express our thanks to him.

References

1. S.M. de Oliveira, P.M.C. Oliveira, D. Stauffer, S.M. de Oliveira: Evolution, Money, War, and Computers. Teubner Verlag (1999)
2. T. Penna: J. Stat. Phys. **72** 1629 (1995)
3. D. Makowiec, J. Dabkowski, M. Groth: Physica A **273** 169 (1999)
4. <http://www.prb.org/>: Population reference bureau (2003)
5. <http://www.mortality.org/>: The human mortality database (2003)
6. Dietrich Stauffer – private correspondence
7. J. Dabkowski, M. Groth, D. Makowiec: Acta Phys. Polonica. **31** 1027 (2000).
8. S.M. de Oliveira, P.M.C de Oliveira, J.S.Sa. Martins: Int. J. Mod. Phys. C15 (2003)
9. J.S. Martins, S. Cebrat: Theory in Biosciences **119**, 156 (2000)